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MONKEYS AS A SOURCE OF VIRAL DISEASES IN MAN

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16. Abstract Under institutional scientific-research conditions, during contact with monkeys or their tissues, there is a danger of infection of the associates by simian viruses which are pathogenic to man. Presented in this paper is information on these stimulants.			
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MONKEYS AS A SOURCE OF VIRAL DISEASES IN MAN

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Under institutional scientific-research conditions, during contact with /138** monkeys or their tissues, there is a danger of infection of the associates by simian viruses which are pathogenic to man. Presented below is information on these stimulants.

Simian herpes virus (virus B). It was first isolated in 1933 from the brain of an associate who died after a monkey bite [20]. It is related to the Herpesviridae family, the subfamily Alphaherpesvirinae, and has common antigens with the herpes virus in man and with herpes in swine (Pseudorabies). The virus is replicated with a cytopathic effect in cell cultures of kidneys of monkeys, man, rabbits, pigs, and lambs, in the amniotic cells of man, in the fibroblasts of chick embryos and in various cell lines of human origin Her-2, HeLa, FL, KB.

With infection of chick embryos on the chorion-allantois membrane, they perished on the 4-6th day, and the virus is found in the body of the embryo.

Under natural conditions, rhesus monkeys (*Macaca mulatta*) may contract virus B. This illness in them is reminiscent of herpes in man. Blisters /139 appear on the tongue, lips and sometimes also on other parts of the body. These blisters subsequently turn to ulcerations with a yellowish-grey necrotic coating. Often conjunctivitis and rhinitis are observed at the same time. The overall condition of the animals remains without any notable changes [26]. Cases have been described in which the illness occurred with giganto-cellular pneumonia [39].

Most of the animals develop changes in the medulla oblongata and pons

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varolii. Evidently, sometimes the virus is retained for a long time in the ganglion of the trigeminal nerve. Cases have been described in which it was found there 6 months after clinical recovers [43]. The study performed by the author of the brain and spinal cord of 75 *Macaca mulatta* monkeys with changes of uncertain ethiology lead to the isolation of virus B in 2 cases. With cultivation of the kidney cells of 6,152 monkeys, the virus was found in 3 cases [2].

Monkeys of different species may be easily infected by the herpes virus from rhesus monkeys, and in these cases the infection may take on a severe form. Cases of the illness with lethal outcome were observed in *Macaca fascicularis* (*cynomolgus*) and in *Macaca radiata*. In the latter, the infection occurred as the hemorrhagic interstitial pneumonia with rhinitis and conjunctivitis [17].

An illness clinically- similar to the natural infection may be reproduced in rhesus monkeys with the introduction of the virus in the mucous membrane of the tongue [26]. Infection in the brain does not always lead to development of the disease.

Of the laboratory animals, the most susceptible were Syrian hamsters, cottonfield rats and rabbits. With any method of inoculation of the virus in rabbits, their nervous system is afflicted [38]. With intracutaneous administration of the virus, an ulceration appears in this area, then paralysis develops. Death usually ensues on the 10-12th day after infection.

There have been over 20 cases described of humans contracting simian herpes [25]. In most cases the process took place as ascending encephalomyelitis with paralysis and change in sensitivity, and only as an exception did it bear a descending character. As a rule, the illness had a lethal outcome.

Only singular cases of recovery are known. The paralysis in this case diminishes, but there is stable paresis, tremor, nystagmus, and loss of skin sensitivity in places [45].

There are indications that it is possible for humans to be long-term asymptomatic carriers of the virus. A case of encephalomyelitis 10 years after contact with monkeys has been described. In this case, skin afflictions appear along the course of the trigeminal nerve, similar to those which occur with zoster [shingles]. The simian herpes virus was isolated from a skin scraping taken at the place of affliction [18].

A natural reservoir of the virus are *Macaca mulatta* monkeys. Antibodies to the virus were also found in other species of the genus *Macaca* and in green African marmosets [3, 16]. However, most probably this was the result of their becoming infected not in nature, but under conditions of captivity as a result of contact with *M. mulatta*. The spread of the virus among monkeys evidently occurs most often through food contaminated with infected saliva, and more rarely--through a bite. The case history of people who have become ill usually includes a monkey bite. However, in a number of cases the contamination evidently occurred through infected saliva which got into a scrape on the skin, or into the eye, nose or mouth [35]. A case of human contamination upon contact with a cell culture from monkey kidneys has been described [15].

Simian pox virus. In 1959 among the monkeys in vivariums in Denmark and the USA there were observed outbreaks of an illness caused by a previously unknown stimulant, which was called the simian pox virus [29, 37]. It is related to the family Poxviridae, genus Orthopoxvirus and has common antigens with the viruses of natural smallpox, vaccines and cow pox. The virus multiplies on the chorion-allantoic membrane of chick embryos. It is also

replicated in primary cultures of kidney cells of monkeys, rabbits, guinea pigs, cattle, and pigs, in the cells of chick embryos, in the human amnion, and inoculated cultures of simian kidney cells, HeLa, the lungs, the thyroid gland, and amnion of man. The afflicted cells in the culture become rounded. The virus remains in the cells and is practically not isolated /140 into the culture medium.

A petechial rash appears on the face, body, extremities and tail of the afflicted monkeys. Then papules, vesicles and pustules appear. The rash is most intensive on the palms and soles of the feet. Then the varioles become covered with a reddish-brown crust, which falls off on the 7-10th day, leaving a scar. The overall state of the monkeys in most cases is not disrupted [29]. Often the infection occurs inapparently, with the virus being found in the renal tissue of the animals. During the epizooty which took place in the Rotterdam zoo, the primary changes in the monkeys were localized in the kidneys in the form of interstitial nephritis [23].

The rhesus macaques and cynomolgus are susceptible to experimental infection with the pox virus. South American broad-nosed monkeys (*Cebus apella* and others) are less sensitive [34].

Rabbits are highly susceptible to infection with the simian pox virus in the brain, on the cornea, and intratesticularly. With infection of scarified skin, papulose-pustulose afflictions develop. Adult mice perish only with introduction of the virus into the brain, and sucklings--also after intranasal infection [37].

The illnesses caused by the simian pox virus in man are indiscernible from natural pox by their clinical picture [1, 19]. The prodromal period lasts for 2-5 days. The temperature becomes elevated, weakness, headache, and sometimes pain in the throat occur. Usually after 3-4 days a rash breaks out over the entire body with predominant affliction of the face and extremities.

As a rule, the process involves the palms and soles of the feet. The rash passes through stages of spots, vesicles, pustules, scab formation, and scar formation. The elements of the rash may have a hemorrhagic character. A case has been described with development of keratitis, after which a leukoma was formed. Lethality comprises around 17%.

All known outbreaks of simian pox occurred among animals in captivity. Macaques, chimpanzees, gorillas, orangutans, gibbons, African green marmosets, marmosets, squirrel monkeys, as well as anteaters [5] were all afflicted. Under natural conditions, the antibodies to this virus in monekeys are rarely found and in low titers [19]. It is possible that monkeys are not a reservoir for this infection.

Cases of simian pox in humans were recorded in various countries of Africa--Zaire, Liberia, Nigeria, Sierra-Leone, Ivory Coast. The infection does not have a tendency toward epidemic spread. Only singular cases of transmission of the disease within families have been described [5]. The sources and means of infecting humans are unclear. There is a report of a case of illness by a child contracting simian pox after he was bitten by a monkey—a chimpanzee [33].

Yaba virus. This is a member of the family Poxviridae, and has common antigens with the Tana pox virus. It multiplies in primary cell cultures of rhesus monkey kidneys, inoculated renal cells of the human embryo (MA-10), human amnion (HuF), and monkey kidneys (LLCMK₂, JINET, BS-C-1). Hyperplastic tumor-like foci appear on the cell layer [48].

On the chorion-allantoic membrane of 10-11 day old chick embryos the virus causes formation of tumors 1 cm in diameter after 5 days [41]. The laboratory animals are not susceptible to infection.

Only one epizooty has been described caused by Yaba virus. It took place at the primatological center in Nigeria in 1957 [9]. The animals there were kept in open-air cages. The epizooty involved the Asiatic rhesus monkeys. The African monkeys were resistant: only 1 baboon became ill.

The illness in monkeys began with the appearance of small reddish papules on the face, ears, hands or feet. These were singular or multiple, and after several weeks reached 2-4 cm in diameter. These tumors consisted primarily of histocytes, did not have capsules and were fixed on the underlying tissues. The tumoral cells particularly infiltrated the muscle tissue. Metastasis was not observed. The general state of the animals was not disrupted. Then the tumoral cells degenerated and the nodules often became ulcerated. After several months the tumors dissolved, and granulation tissue /141 grew in their place.

It was found that rhesus monkeys in nature do not become infected with the Yaba virus. Of the Asiatic monkeys, *Macaca fascicularis* and *Macaca radiata* had antibodies to the virus. The intensive circulation of the virus was found among African green marmosets [42].

People become infected through scratches inflicted by the monkeys. After 5-7 days a subcutaneous nodule appears in this area, which subsequently increases in size, reaching a diameter of 2 cm. After 3-4 weeks the nodules become dissolved. A case of infection through an infected needle was described, in which the tumor developed at the place of inoculation of the virus after 4 months [24].

Tanapox virus (Tanapox virus) is a member of the family Poxviridae. It multiplies in primary cell cultures of human and rhesus monkey amnion, in cells of the human thyroid gland, in the Wi-38 diploid cells of the human lung, in re-inoculated cultures of monkey kidney cells--Vero, CV-1, BS-C-1 and others. The virus did not exhibit the capacity to multiply in bird

embryos. Of the laboratory animals, only rabbits of certain breeds turned out to be susceptible: with infection, small red papules appeared on their skin.

Outbreaks of Tanapox among monkeys occurred in 1966 in 3 primatological centers in the USA, when macaques became ill. They developed a few flat papules 1-3mm in height and up to 10mm in diameter on their skin. The center of the papules was slightly depressed, with a crust. The skin around the papules was slightly hyperemic. The papules consisted of epidermal cells [32]. The illness had a mild course. The skin afflictions disappeared after 4-6 weeks.

Antibodies to Tanapox virus were found in 15-20% of the green marmosets of Kenya and Ethiopia [30], as well as in the macaques of Malaysia.

Outbreaks of Tanapox virus among humans occurred in Kenya in the Tana River valley in 1957 and 1962. During the epidemic in 1963, several hundred people contracted the disease, primarily school children. Evidently, this infection is rather widespread in Kenya. Antibodies were found in a significant number of residents [8]. The infection of humans evidently occurs through mosquito bites.

During the outbreak in 1966 among the monkeys in the primatological centers in the USA, 5 associates and 2 animal traders who came in contact with the monkeys became ill. In 1 case the variole appeared at the point of a scratch on the skin. Evidently this is the usual means of infection with direct contact with sick animals. The incubation period in man lasts about 3 days. Singular papules, red in color and with diameter of 2.5 cm appear on the open parts of the body. These are surrounded by a zone of inflammation. Then the center of the papules turns white and becomes covered with a crust. The body temperature remains normal or is insignificantly elevated. Sometimes a painful enlargement of the lymphatic glands, pain in

the back, and strong headache are noted. The skin formations disappear usually in 2 months [30, 32].

Marburg disease virus. This disease, which has come to be called Marburg's disease, was first recorded in 1967 in Marburg, Frankfurt (FRG) and Belgrad (Yugoslavia) among workers removing and processing kidneys from green marmosets from Uganda, as well as among persons coming into contact with those who became ill. A total of 31 persons became ill, and 7 of them died. The means through which the infection entered was damaged skin or mucous membranes. The incubation continued for 4-7 days. The patients exhibited an elevated temperature, headache, vomiting, and impaired consciousness. Objectively, conjunctivitis, enlargement of the lymph glands, exanthema in the form of red-colored papules on the skin, enanthema, and hemorrhagic diathesis were noted. Death ensued on the 8-16th day. In non-lethal cases, recovery began on the 15th day and proceeded slowly [31]. Those who recovered were virus carriers for a long time. In one case, the virus was found in a conjunctiva wash 80 days from the start of the illness. In another, it was isolated from the sperm on the 83rd day. A case of infection resulting from sexual contact with a convalescing patient was described.

In those who died, the changes occurred in the form of hemorrhagic diathesis and necroses in various organs, and were particularly expressed /142 in the liver and the brain [10].

The Marburg's disease virus is reminiscent of rabdoviruses by its structure. We were able to adapt it to a cell culture of human amnion, to lines of renal cells from green marmosets AH-1 and Vero, to lines of renal cells from the Syrian hamster VNK-21, clone CCI-10. Multiplication of the virus is accompanied by a mildly expressed destruction of the cell layer [27].

With primary infection of Vero cells with a diagnostic purpose, the viral antigen may be isolated on the 2-3rd day with the aid of immunofluorescence [47].

The virus is pathogenic to guinea pigs, in whom it is generally the liver which is afflicted. After a series of passages through the organism of guinea pigs and monkeys, the virus took on a pathogenic character for Syrian hamsters, and then for newborn mice (strain F). The data regarding the illnesses caused by Marburg's virus in monkeys under natural conditions are absent. With experimental infection of green marmosets, rhesus monkeys and squirrel monkeys parenterally, they get sick and soon die.

After an outbreak of Marburg's disease in Europe in 1967, the disease was registered in 2 tourists and a nurse in Rhodesia in 1975 [28], and then 1 primary and 2 secondary cases in Kenya in 1980 [40, 44]. Two of those who became ill died. Antibodies to the Marburg's disease virus were found in Africa in green marmosets and hussar-monkeys. We have not excluded the possibility that a reservoir of the virus are not monkeys, but some other animals, most probably rodents, and that arthropods are its carriers [6].

Yellow fever. The yellow fever virus is related to the genus Flavivirus, family Togaviridae.

The virus causes encephalitis in baby mice and in guinea pigs. In European hedgehogs with extraneural infection, a necrotic process develops in the liver. An effective method of isolating the virus is intrathoracic infection of mosquitos, with subsequent passage through the brain of baby mice [7].

Endemic foci of yellow fever are found in the tropical and subtropical regions of Africa, South and Central America. The main reservoir of the

virus in Africa are African green marmosets. Evidently, the infection is also widespread among baboons, monkeys of the genus *Colobus* and half-monkeys of the genus *Rottos*. In South America, howlers, squirrel monkeys, marmosets, spider monkeys, capuchins, and other species are afflicted. Infected howlers, marmosets and squirrel monkeys usually die with the appearance of hepatitis. Spider monkeys undergo a milder course of the disease, while capuchins most often experience inapparent infection. In the forests of South America, the infection is spread by the mosquitos *Haemagogus spegazzini* and *Aedes leucocoleanus*. In the forests of Africa it is spread by *Aedes africanus*, *A. simpsoni*, *A. furcifer*, *A. Taycanus*, *A. simpsoni*, *A. furcifer*, *A. Taylori*, *A. diceromyia*, *A. luteocephalus*, *A. neoaffricanus*, *A. aedimorphus* [11-14, 21, 36]. There are reports of the isolation of the virus in Africa from the ticks *Amblyomma variegatum* [22, 39]. It is as yet difficult to judge the significance of this fact.

Among monkeys there are often observed severe episodes of yellow fever with high mortality rate. The disease rate in humans in endemic (jungle) centers is low. However, such a focus may be the start of an epidemic (urban) form of yellow fever with a very high disease rate. Monkeys do not participate in this process but the virus is transmitted from man to man by the mosquito: *Aedes aegypti*. Such epidemics may occur not only in population centers of hot countries, but also in the summer period in regions with moderate climate and in the presence of the proper carrier (Italy, France, Portugal, USA).

In man, after a 2-6 day incubation period, the temperature becomes elevated and the pulse sharply quickens. The patients complain of pain in the back and legs, and headache. On the 3rd day the temperature usually goes down and the patients feel a little better. However, soon the temperature again goes up, and this time is combined with bradycardia. The patients suffer

from severe nausea and vomiting, with simultaneous pains in the epigastral region. The vomit mass and stools are usually black in color. Jaundice, as a rule, is of moderate intensity, and may be absent altogether. Recovery /143 is quick, without residual effects.

The mortality rate with yellow fever comprises over 40%. Death usually ensues on the 2nd or 4-7th days of the illness. The basic changes consist of fatty regeneration of the liver and kidney tissues, destruction of the liver parenchyma and hemorrhage in various organs [11].

Kiasanuri forest disease. The stimulant is related to the genus Flavivirus of the family Togaviridae. It has common antigens with the viruses of tick-borne encephalitis, Omsk hemorrhagic fever, and Scottish encephalitis in sheep. The virus multiplies in cultures of kidney cells from monkeys, Syrian hamsters and guinea pigs, as well as in chick embryo cells without appearance of cytopathic changes. Mice, baby Syrian hamsters and palm squirrels are sensitive to the infection by the virus.

The disease in humans is known since 1955, when an epidemic broke out in the region of the Kiasanuri forest (India). It occurs with hemorrhagic syndrome, but without rash and hematuria. The incubation period comprises 5-8 days. The temperature goes up rapidly to a high number, strong headache is noted, as well as pain in the lower back and extremities. The patients are apathetic, their speech is inhibited. On the 3-4th day of the illness the condition deteriorates. The patients suffer from nausea, vomiting and diarrhea. The sclera and conjunctiva are hyperemized, and sensitivity to light is noted. Populo-vascular rash on the soft palate is characteristic. Bleeding of the gums appears, there are nosebleeds and gastro-intestinal bleeding, and spitting up blood. On the 7-14th day of the illness the temperature goes down to normal, but the hemorrhaging continues for several more days.

Weakness and pain in the extremities remains. The convalescent period lasts for 1-2 months, but recovery is complete.

Sometimes after normalization of the temperature, most often on the 9-12th day of the illness, a second wave of the illness ensues, lasting up to 7 days. It occurs with disruption in the activity of the central nervous system [4].

The mortality rate with the Kiasanrui forest disease comprises around 15%, reaching up to 28% with certain outbreaks [46]. Death usually ensues on the 7-9th day of the illness. The basic afflictions are concentrated in the liver and kidneys.

Cases of Kiasanuri forest disease in humans are usually preceded by massdeath of monkeys in a given area--macaques (*Macaca radiata*) and langurs (*Presbytus entellus*). In the endemic region the antibodies were found in 8% of the macaques and 20% of the langurs. However, generally the virus circulates among rodents--*Tatera indica hardwickei*, *Suncus murinus*, *Rattus wrongtoni*, and palm squirrels. The antibodies were found also in birds--the grey tropical duck and woodpecker [4]. The reservoir and carrier of the virus are the ticks *Haemaphysalis spinigera* and *Haemaphysalis turanicus*. After the epidemic of 1955, the infection has a tendency to spread to regions adjoining the Kiasanuri forest.

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